ABSTRACT

Regulation of expression of CTL activity by macrophage migration inhibitory factor (MIF) is disclosed. In a mouse model using the EL4 tumor, cultured splenocytes from tumor-primed mice secrete high levels of MIF following antigen stimulation *in vitro*. Parallel splenocytes treated with neutralizing anti-MIF mAb showed a significant increase in CTL response against tumor cells compared to control mAb-treated cultures, with elevated expression of IFNγ. Histology of tumors from anti-MIF treated animals showed increases in infiltration of both $CD4^+$ and $CD8^+$ T cells, as well as apoptotic tumor cells, consistent with observed augmentation of CTL activity *in vivo* by anti-MIF, which was associated with enhanced expression of the common γ_c chain of the IL-2 receptor that mediates$CD8^+$T cell survival. $CD8^+$ cells of anti-MIF treated tumor-bearing mice showed increased migration into tumors of control mice. Methods for enhancing a CTL response by inhibition of MIF are disclosed.